

Home Refinishing, Lead Paint, and Infant Blood Lead Levels

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Abstract: We measured the blood lead levels of 249 infants semi-annually from birth to two years of age; we sampled the home paint and recorded any recent home refinishing activity. Mean blood lead from birth to age 2 years did not vary systematically with age but did correlate significantly with the amount of lead in the indoor paint ($p < .01$). Refinishing activity in homes with high lead paint was associated with elevations of blood lead averaging 69 per cent. (*Am J Public Health* 1985; 75:403-404.)

Introduction

Evidence is accumulating that current community exposure to lead has detectable adverse effects in children.¹⁻³ Many children with lead poisoning have ingested lead-containing paint chips, pulled off walls and window sills. New parents, motivated to minimize the hazards of lead, frequently resurface or repair their dwellings. Previously, we have reported on the relationship of resurfacing during pregnancy on cord blood lead levels.⁴ We report now the relationship of this activity to the lead levels of infants.

Methods

The base population consisted of 11,837 consecutive births at the Boston Hospital for Women, between April 1979 and April 1981. We identified 1,207 children in the highest, lowest, and middle deciles of cord blood lead. These babies were then eligible for the follow-up sample if:

- the family lived within a radius of about 12 miles from the hospital, but outside certain public housing projects and specific inner-city areas which were judged to be unsafe for our field personnel (365 excluded);
- hospital consent forms were signed in English (65 excluded);
- the child had no serious medical condition which required a hospital stay of more than two weeks (38 excluded). In addition, 42 families were moving, 85 were unreachable, and 167 refused to participate. Compared with the subjects who were eligible by virtue of their lead level but not enrolled, the enrolled mothers were more likely to be older, married, college educated, White, and employed. Although enrolled mothers consumed more alcohol, they did not differ from the eligible non-enrolled mothers in general health, reproductive history, course of this pregnancy, or birth status.

We enrolled 249 infants nearly equally drawn from three distinct cord blood lead categories (< 3 , $6-7$, and > 10 ug/dl). The participating mothers had a mean age of 29 years, and a mean schooling of 15 years. Eighty-seven per cent were White, 68 per cent were working at the time of conception, and 77 per cent had some formal childbirth

preparation. A total of 204 completed the two-year study, resulting in an annual attrition rate of less than 10 per cent.

At ages 1, 6, 18, and 24 months, paint lead was measured in each child's residence, both by X-ray fluorescence with a PGT Model XE-3 (Princeton Gamma Tech, Princeton NJ) at three interior sites in the home, and by atomic absorption of a digested sample taken from any indoor spot where paint could be removed without disturbing the surface excessively. All sites chosen were accessible to the children and included a windowsill and two walls. Further details have been reported elsewhere.⁵ Complete paint lead data were obtained for only 91 of the infants because of lack of easily-removable paint layers, and the adverse effects of cold weather on the reliable performance of the portable x-ray unit.

The readings at the three sites were averaged. To combine the two types of paint data, we constructed an ordinal scale in which a score of 0 was $< 1/2$ per cent Pb by wet chemistry or < 0.4 mg/sq cm by X-ray. A score of 10 was assigned to any value equal or greater than 10 per cent or mg/sq cm. These categories of paint lead are not evenly populated but serve to rank potential exposure.

The parents were also asked if during the last six months there had been any refinishing activity in the home, i.e., sanding, scraping, or painting. A yes or no was recorded. It was not determined whether the refinishing corresponded to the same surfaces for which lead paint data was obtained.

Details of the analytical procedure for blood lead (PbB) which involve anodic stripping voltammetry have been reported previously.⁶ At ages 6, 12, 18, and 24 months, a capillary blood sample, collected by a trained technician, was assayed in duplicate or triplicate with an ESA model 3010 anodic-stripping voltammeter utilizing an exchange reagent.⁶ There was no trend in PbB with age in this population⁷ so for descriptive purposes the children could be ranked into four categories of PbB, using the mean of the available repeated blood examples from each child (Table 1).

Results

Paint lead levels show a monotonic increase across the mean PbB categories, but the mean paint lead levels are not very different in the extreme categories (Table 1). Even for children in the lowest quartile category, paint lead levels were excessive by local statute (more than 1.5 per cent or mg/sq cm) in 38 per cent of the homes.

Parents of children with elevated lead levels reported having done some refinishing during the study more frequently than did parents of other children (Table 1). Indeed, the mean change in PbB over the six-month intervals (ages 6 to 12, 12 to 18, and 18 to 24 months) when there was no refinishing was -0.27 ug/dl (SE = 0.29, 438 intervals), not significantly different from zero (Table 2). For the 150 intervals when refinishing was reported, however, the change was significantly larger ($+1.40$ ug/dl, SE = .72, $p = .03$).

This change in PbB associated with refinishing varied with the lead content of the paint. Although resurfacing

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⁷The average difference between duplicates was 1 ug/dl and was 3.5 or more in 10 per cent of the pairs.

TABLE 1—Residential Characteristics of Infants in Quartile Categories of Blood Lead, Mean (Std Error)

Environmental Variable	Mean Blood Lead Category ^a				p
	Low <3.7	Mid-Low 3.7–6.0	Mid-High 6.1–8.7	High 8.8+	
Number of Children					
Total	74	59	59	56	
With paint data	22	24	25	20	
Paint Pb (mg/sq cm)	1.8 (.6)	2.3 (.5)	2.6 (.4)	2.8 (.4)	0.001.b
Ever Refinish (% yes) during entire study	56	61	78	79	0.001.b

^aMean of available ages 6, 12, 18 and 24 month PbB ($\mu\text{g/dl}$).^bChi-squared test for linear trend.

TABLE 2—Recent Resurfacing or Refinishing and Infant Blood Lead Level

Resurfacing Reported This Interval	N	Change in Blood Lead Over 6-Month Interval		
		MEAN ($\mu\text{g/dl}$)	STD ERR	% RISE
No	438	-0.3	0.3	-4
Yes	150	+1.4	0.7	+20
Paint Lead				
Unknown	106	+0.9	0.8	+13
<1.2%	19	+1.1	1.4	+16
1.2–>3%	12	+2.8	2.3	+40
>3%	13	+4.8	2.2	+69

information was obtained from every family at nearly every interval, paint data were usually missing. There were only 44 intervals with refinishing and known paint lead scores. In the 13 of these situations of refinishing with documented very high lead paint (scored > 4.0), the change in PbB was 4.8 $\mu\text{g/dl}$ (SE = 2.2, $p = .02$), a rise of 69 per cent. In contrast, for the 19 children in whose homes there was refinishing but had low lead paint (score < 1.3), the change in PbB was only 1.1 (SE = 1.4, $p = .4$).

Discussion

We did not record the exact dates of any refinishing during the six-month period, nor did we record the actual methods of resurfacing used (heat gun, scrapping, liquid paint remover, blast abrasion, or power sander), although this may have influenced exposure. We do not know if the sites where we assayed the paint were representative of the entire home or of where the resurfacing occurred. The age of the dwellings (a useful surrogate for paint lead), the presence of outdoor paint lead and other high-dose sources of lead to

the child were unmeasured. Given these gaps in the data, the underlying relationships between paint lead, refinishing, and rises in infant blood lead should be interpreted with caution.

Much of the refinishing may have been motivated to lower the hazard to the child from lead paint. However, this activity could have had the undesired effect of increasing, at least temporarily, the child's PbB. Infants residing where lead paint is being resurfaced (as typically performed) may be at special risk of increased lead exposure.

REFERENCES

1. Needleman H, Rabinowitz M, Leviton A, Linn S, Schoenbaum S: Relationship between prenatal lead exposure and congenital anomalies. *JAMA* 1984; 251:2956–2959.
2. Bellinger D, Needleman H: Low level lead exposure and psychological deficit in children. *Adv Devel Behav Ped* 1982; 3:1–49.
3. Needleman H, Gunnoe C, Leviton A, Reed R, Peresie H, Maher C, Barrett P: Deficits in psychological and classroom performance of children with elevated dentine lead levels. *N Engl J Med* 1979; 300:289–295.
4. Rutter M: Raised lead levels and impaired cognitive/behavioral functioning. *Dev Med Child Neurol* 1980; (Suppl)42:1–26.
5. Piomelli S, Seaman C, Zullo D, Curran A, Davidow B: Threshold for lead damage to heme synthesis in urban children. *Proc Nat Acad Sci USA* 1982; 79:3335–3339.
6. Rabinowitz M, Needleman H: Demographic, medical, and environmental factors related to cord blood lead. *Biol Trace Element Res* 1984; 6:57–67.
7. Rabinowitz M, Needleman H, Leviton A: Variability of blood lead concentrations during normal infancy. *Arch Environ Health* 1984; 39, 74–77.

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